The role of the milking machine in the aetiology and epidemiology of bovine mastitis

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Abstract

Mastitis is an inflammation of the mammary gland that causes major economic losses in developed dairy countries.

A great deal of research has been directed toward the identification of machine factors related to mastitis. The milking machine has little effect on the new mastitis infection rate if it is installed, operated, and functions according to internationally recognised standards. Its role in causing mastitis is often overestimated. It has proven difficult to produce mastitis experimentally solely by altering machine functions within accepted parameters.

Although not a direct prerequisite for mastitis, the milking machine has significant effects upon the aetiology and epidemiology of mastitis. These effects may operate directly by increasing the new intramammary infection rate, or indirectly by increasing the risk of exposure of the mammary gland to mastitis-causing organisms, and reducing disease resistance in the cow.

There are four major identifiable ways in which milking machine can influence the development and severity of bovine mastitis, the physical transport of mastitis-causing organisms between quarters and cows, causing damage to the teat end, increasing the risk of mastitis causing organisms penetrating the teat duct and increasing the colonisation of the teat canal with mastitis-causing organisms. However, any one property, mechanical or operational, may cause an increase in the new intramammary infection rate. The commonest management factors associated with operational properties of the milking machine and its influence on the new intramammary infection rate are over-milking and under-milking.

In general milking machines have little influence on mastitis occurrence on the farm if the milking equipment is suited to the facilities available, properly installed, functioning and regularly maintained, periodically tested, and deficiencies corrected.

Introduction

Mastitis is an inflammation of the mammary gland. It is a major cause of economic losses to the dairy industry in developed dairy countries (Gill et al., 1990; McInerney et al., 1992; Pyorala, 1992; DeGraves and Fetrow, 1993; Allore and Erb, 1998; Hortet and Seegers, 1998; Allore and Erb, 1999; Seegers et al., 2003). The most common cause of mastitis is infection with mastitis-causing organisms, which enter the gland via the teat canal, and overcome the defence mechanisms of the udder.

A great deal of research has been directed toward the identification of machine factors related to mastitis. The milking machine (MM) has little effect on the new infection rate if properly operated and functioning according to the manufacturer's specifications and internationally-accepted standards. Even though the milking system becomes the focus of many herd udder health investigations, there is a little evidence that machine factors are of primary importance in most problem herds. It has often proven difficult to produce mastitis experimentally solely by altering milking machine functions provided they remain within accepted parameters. The authors believe its role in causing mastitis problems is frequently overestimated; a conclusion that has been reached by some other authors (Kirkbride and Erhart, 1969; O'Shea et al., 1984; Osteras and Lund, 1988: Spencer, 1988).

Although not a direct prerequisite for mastitis, the MM has significant effects upon the aetiology and epidemiology of mastitis. These effects may operate directly by increasing the new intramammary infection (IMI) rate, or indirectly by increasing exposure to mastitis-causing organisms and by reducing disease resistance in the animal.

There are four major identifiable ways in which MM can influence the development and severity of bovine mastitis:

1. Physical transport of mastitis-causing organisms between quarters and cows,
2. Causing damage to the teat end,
3. Increasing the risk of mastitis causing organisms penetrating the teat canal and
4. Increasing colonisation of the teat canal with mastitis-causing organisms.

The relative importance of these categories varies markedly between the organisms because of their differing sources and properties. However, the MM can affect mastitis incidence on dairy farms under field conditions. It is crucial that these effects of the MM on mastitis are well understood by farmers, dairy advisors, MM testers and veterinarians, particularly those involved in mastitis solving problems, who should be able to help identify and rank the importance of possible problem areas.

The role of the milking machine in assisting the transfer of mastitis-causing organisms between cows or between quarters.

Within any herd, there is a mixture of cows, some with healthy udders and others with either clinical or sub-clinical IMI. The infected cows may excrete mastitis-causing organisms in their milk. Additionally, cows' teat surfaces may become contaminated with mastitis-causing organisms acquired from the environment between milkings (e.g. coliforms, Strep. uberis) or they may be present in the teat sores and other skin lesions on the udder (e.g. Staph. aureus, Strep. dysgalactiae, Strep. agalactiae). The milking process therefore offers many opportunities for mastitis-causing organisms to be physically transmitted from cow to cow or from one quarter to other quarters in the same animal (McDonald, 1969; Grindal, 1988; Spencer, 1988; Woolford, 1995; Edmondson, 2001; Dodd, 2003).

Movement of infected milk between liners

During milking, vacuum fluctuations in conventional claws may result in moving milk between liners (reflux or backflow). If the cow being milked has one or more infected quarters, this process transfers mastitis-causing organisms to the surfaces of other teats. However, cross-contamination does not necessarily lead to new IMI, as despite high levels of challenge by mastitis-causing organisms, the new IMI rates have sometimes remained low

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(O'Shea et al., 1984; Mein et al., 2004). Consequently, MM modifications designed to prevent inter-quarter transfer may usefully contribute to mastitis control. The aim is to prevent cross-contamination without redesign or replacement of the MM itself. Examples of this are the multi-valve or ball claw such as the Ambic Hydraflow Claw and the Alex Harvey Industries (AHI) (now Waikato Milking Systems) Isolator Claw. Another modification is the use of large capacity claw-pieces, aiming to limit cross contamination by removing the milk rapidly from the short milk tubes (Griffin et al., 1988; Spencer, 1988; Edmondson, 2001).

Transfer of Infected Milk from Cow to Cow by Using the Same Cluster

After an infected cow has been milked, contaminated liner surfaces carry mastitis-causing organisms from the teat surface and milk of the infected animal to the next animal when the machine is applied. This may continue for the next five to eight cows after milking an infected animal (Phillips, 1982; Holdaway, 1990; Edmondson, 2001). Segregation of the clinically infected cow, exclusion of her milk from the main supply and milking her after the main mob are all recommended to both help reduce the risk of cross infection and to simplify milking management. Segregation is also commonly recommended for all freshly calved cows and during periods of high mastitis incidence in the herd. The difficulties of recognising affected cows are well-known, however. In some cases, clots are present only in the foremilk. In cases where the clots have resulted from teat canal infection, it may not be necessary to discard all the milk. Conversely, mastitis milk may not contain clots or the clots may be so small that they are invisible to the naked eye (for example with early coliform mastitis; Jones and Ohnstad, 2002). In-line mastitis filters or detectors can operate in the long milk tube between the cluster and the milk line, jar or meter, but are not normally examined until the cow has been milked and, again, do not detect all cases. Consequently, it is virtually impossible to prevent all transfer in practice.

The milking machine as an aid in the multiplication of mastitis-causing organisms at the teat end.

Mastitis risk is, theoretically, a 'numbers game', inasmuch as greater numbers of mastitis-causing organisms near the teat end increase the risk of infections occurring (Grindal, 1988; Mein, 2004). The major factor influencing new IMI is exposure of the teat orifice and canal to mastitis-causing organisms in conjunction with machine or weather induced teat end damage (McDonald, 1969; Thompson, 1977; Edmondson, 2001). Milking machines can influence teat end contamination by (a) modifying conditions at the teat end and (b) causing teat-end lesions which allow colonisation by mastitis-causing organisms, particularly with Staph. aureus and Strep. dysgalactiae, to occur more readily. Teat-end lesions associated with over-milking, high milking vacuum, worn liners and long-term inadequate massage (i.e. a restricted 'D' phase in pulsation) are referred to as teat orifice 'eversion' or 'hyperkeratosis' where the teat canal looks like it is being pulled inside out (Edmondson, 2001). 'Haemorrhagic blisters' on teat ends develop as an early sign of high milking vacuum, obvious on non-coloured teats. 'Teat chapping' and 'black pock' lesions have been associated with improperly operating MM's but are also greatly influenced by inclement weather and failure to use suitable emollients in those circumstances to maintain teat condition. Such skin abnormalities are readily colonised by mastitis-causing organisms, and the risk of new IMI will increase due to the impaired ability of the teat canal to prevent organisms entering the udder (Edmondson, 2001). The regular use of a teat disinfectant with added emollient, such as glycerol, can make a valuable contribution to the maintenance of skin elasticity and to the rapid healing of lesions.

The role of the milking machine in the increase of teat canal penetration by the mastitis-causing organisms

The Impact Theory

Abrupt loss or reduction in milking vacuum may create changes in air movement of sufficient force to move mastitis-causing organisms past the teat canal defences, from the exterior of the teat into the teat sinus. This phenomenon is known as the impact mechanism. Many authors (Thompson, 1977; O'Shea et al., 1984; Grindal, 1988 Griffin et al., 1988; Rogers and Spencer, 1991; Rasmussen et al., 1994; Edmondson, 2001) consider this to be one of the main effects on the new IMI rate. During the normal pulsation cycle, the vacuum at the teat end varies with the opening and closing of the teat liner. On opening of the liner vacuum at the teat end increases through the increase in volume created by the opening liner. This is known as cyclic vacuum fluctuation, and is a consequence of normal pulsation. If, on top of the normal cyclic vacuum fluctuation, there is a sudden drop in vacuum level in the rest of the milking system, impact theory suggests that the vacuum differential can be so great that milk droplets impact against the teat end with sufficient force and speed to pass the teat canal and enter the gland. A new IMI may occur if these droplets contain mastitis-causing organisms. Whether mastitis-causing organisms will cause mastitis or not, depends on the defence mechanisms of the cow, the numbers of invading organisms, and the time of entrance to the gland relative to milking (Rasmussen et al., 1994). If mastitis-causing organisms penetrate into the gland, just prior to cessation of milking when there is little milk left to be removed, it is more likely that they will remain in the udder until the next milking. This will allow time for multiplication and possibly infection to develop (Rasmussen et al., 1994; Rasmussen and Aaerestrup, 1995; Edmondson, 2001). On the other hand, mastitis-causing organisms entering the teat sinus at the beginning of milking are more likely to be, but not necessarily, flushed out during milking (Rasmussen et al., 1994; Rasmussen and Aaerestrup, 1995) as they may be trapped in, for example, damaged tissue (Johnston, 1938).

The frequency of impacts is likely to increase with sudden large air admissions into the cluster, due to liner slips, removal of teat cups, inadequate positioning of the milking unit under the cow and vigorous machine stripping with associated liner slip. Milk droplet impacts can be minimized by avoiding abrupt vacuum loss in the claw that may occur through poor cluster application and removal and liner slip.

The Reverse Pressure Gradients Theory

This relatively 'modern' theory on how mastitis-causing organisms enter the teat started back in about 1938 by Thomas Johnston, whose research, using cut teats and carbon particles, suggests that organisms on the teat end after milking get slowly sucked in through the teat canal after the cluster is removed, or when the cow moves, due to the intermittent pressure on teats from the legs, provided the teat canal is open (Johnston, 1938). Later, Craven (1985) noticed that organisms are carried upwards through a narrow tube on rising fat globules. Improved pressure transducer technology in the early 1990s helped to identify the reverse pressure gradient (RPG) across the teat canal as a physical mechanism capable of facilitating the movement of mastitis-causing organisms through or deeper into the canal, or even into the teat cistern (Rasmussen et al., 1994; Rasmussen and Aaerestrup, 1995). RPGs are found whenever the teat is squeezed and released; such as by bending it over for example when the cow lies down or by applying and removing clusters. Whether organisms can enter the teat at this time will depend on the presence of mastitis-causing organisms at the teat end and whether the teat canal is open; a situation made more likely when teat damage or swelling prevents normal closure. Transfer is theoretically possible with, for example, careless cluster removal or liner slip. This suggested mechanism could explain entry of the environmental mastitis-causing organisms as they do not colonise the teat canal and skin, and therefore need an active force in order to penetrate the canal. The RPG mechanism is one way to explain how the environmental mastitis-causing organisms enter the teat sinus at a time when no MM contacts the cow (after drying off and before calving), but infection with environmental organisms is a relatively common problem.

The role of the milking machine in modifying the teat or intramammary environment to enhance infection by mastitis-causing organisms or to impair host defences.

Milking machines may adversely affect udder health by damaging or changing the resistance of the cow's first line of defence (i.e. teat skin, teat canal and mucosal tissue) thus rendering it more susceptible to colonisation by mastitis-causing organisms. Teat sores and cracks provide sites where organisms can multiply. They can be painful to the cow, causing her to kick more frequently during milking time, have poor let-down, increase milking time or prevent emptying of the udder, so that increased risk of mastitis is likely. Local pain may lead to neuro-hormonal responses that suppress immune function (e.g. cortisol and bradykinin releases), and increase the likelihood of disease as well as interfere with milk ejection or the 'letdown' process (Bruckmaier and Blum, 1998; Eshraghi et al., 1999; Wellnitz and Bruckmaier, 2001). The effects caused by improper pulsation are covered later. Some of the teat canal changes influenced by the MM are mentioned above (such as teat canal hyperkeratosis, eversion and erosion). The MM can affect keratin production and removal from the teat canal. When the keratin is mechanically removed, the teat canal loses much of its ability to resist invasion by mastitis-causing organisms. During the milking process, some keratin is always removed. This partial removal of the keratin leads to a flushing effect and lowering of the organisms' counts at the teat end and in the teat canal (Lacy-Hulbert and Woolford, 2000). According to Woolford (1995), the amount of keratin removed during the normal milking process is up to 40%. However, this keratin is quickly replaced, and it has been postulated that the removal of small amounts of keratin during milking and the associated removal of bacteria attached to the keratin may be a major part of the teat defences (Grindal, 1988). It is also suggested that removing too little keratin (as in the SV Swinging Vacuum Single-Chambered milking system) or too much (by over-milking etc) may result in insufficient active keratin behind to help defend the udder. In such a situation the teat canal is more susceptible to invasion by the mastitis-causing organisms. This is common if cows are milked with a faulty MM.

Common problems of the milking machine associated with mastitis

In efficient milking systems the losses in vacuum during milk-flow between the teat-end and the milk receiver are minimised and the maximum amount of milk is extracted with minimal levels of liner slippage and minimal stress to the cow. Any milking operation that causes discomfort to the cow whether by faulty equipment or improper techniques may lead to udder injury or mastitis. Many combinations of improper installation, operation, and utilisation of milking equipment may create situations that irritate or damage the teat or udder tissues.
Milking machine factors that are commonly associated with an increased incidence of mastitis follow:

Vacuum level and fluctuations

The basic principle of the MM is to use vacuum to extract milk from the cow. An excessively high vacuum level and vacuum fluctuations may have a harmful effect to the teat end. The teat end vacuum should be at a level and degree of stability compatible with rapid, complete milk extraction and minimal tissue trauma. Lower than optimal values may result from excessive milk line height, restrictions in the milk tubes, excessive vacuum drop across ancillary components, blocked claw air admission holes and excessive air admission through air admission holes or air leaks into the cluster. Increasing the system vacuum level, may result in faster milking time but be offset by higher strip yields, a higher incidence of hyperkeratosis at the teat orifice, and more machine-induced teat congestion and oedema, that conversely may also slow down the milking process.
It is recommended the milking system be designed so that vacuum fluctuations in the receiver are less than 2 kPa during normal operation, and that the nominal operating vacuum following an incident such as during cluster application or fall be restored within 3 seconds. For optimal teat health and reasonable milking speed the vacuum at the teat end is commonly maintained at about 40-42 kPa. The ISO document 5707 suggests a mean claw vacuum of 32-40 kPa during peak milk flow (Rasmussen and Madsen, 1998; Gleeson et al., 2004) as a 'good compromise'

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between milking rate and teat health (ISO, 1996). A higher vacuum can be tolerated with good preparation and minimum cups-on time illustrating the range of opinion on settings that can give equivalent results. (see Table 1)

Table 1. Vacuum level vs. height of milkline above cow platform

Milkline height above cow platform (m)	Vacuum (kPa)
1.8	48
48	46-48
1.4	44-46
1.2	42-44
Lowline	40-42
These settings may be modified slightly depending on cluster design and components	

Under experimental conditions, cows milked with high cyclic and irregular vacuum fluctuations suffered higher udder infection rates than cows milked with minimal vacuum fluctuations (McDonald, 1969; Grindal, 1988). Irregular vacuum fluctuations may cause or be caused by liner slips or drop off and consequently increase the opportunities for new IMI.

Air leaked inadvertently into a system such as may be caused when a cluster is kicked off an udder, because of liner slip, rough cluster removal from the cow and/or a poorly designed or maintained plant can all contribute to severe vacuum fluctuations.

Pulsation failure

Pulsation acts to massage the teats, promote blood circulation and relieve the oedema and teat end congestion caused by exposure to vacuum. Faulty pulsation is a major cause of teat damage. If pulsator ports are dirty or worn the flow of air may be restricted so that the vacuum level between liner and shell is not reduced to zero during the pulsation cycle; thus, the liner does not collapse completely with sufficient pressure on the teat end to massage it properly. Congestion of blood vessels and tissue fluids (oedema) results (Hamann and Mein, 1990). This leads to slower closure of the teat canal and/or hypoxia in the teat tissues (Hamann and Mein, 1990; Mein et al., 2004) increasing the susceptibility to new IMI (Rasmussen and Madsen, 2000; Gleeson et al., 2004). This effect may also be associated with micro-lesions in the teat canal, sinus and skin, rendering them more susceptible to colonisation by mastitis-causing organisms. Appropriate pulsation is important for sufficient teat end massage. Mechanical failure of the pulsator, shortness of the liner barrel, or a liner 'rest' or 'D' phase that is too short are the most common causes of pulsation failure.

Typical recommended pulsation ratios that can be used with safety range from 50% to 65% (Thiel and Mein, 1977). Ideally, one should keep the milking ratio to within range of 60-65% provided the 'D' phase is greater than 20% or 200 milliseconds. Ratios as wide as 70% are available from some equipment manufacturers. These units may milk quicker but require careful supervision to avoid teat congestion (Gleeson et al., 2004). Such wide ratios also are more likely to cause irritation to the animal during milking if malfunctioning occurs and if the 'D' phase is restricted. It should also be noted that the rate of pulsation affects the speed of milking by only a relatively small amount; whereas excessive rates may have adverse effects. Most modern MM's perform well at rates of 50 to 60 pulses per minute if the remainder of the system is operating properly. Faster rates may not provide adequate teat massage or rest under varying vacuum levels, but are sometimes used as a stimulation phase to encourage milk letdown. Some modern machines can widen the ratio during peak milk flow to increase milking speed while keeping the duration of the 'D' phase constant.

Selection and care of teat cups and liners

Liners (Inflations) must fit the teat cup, claw and teat. Compatibility tables are available from MM and liner manufacturers. Liners are usually made of synthetic rubber that has a slightly porous surface that becomes progressively worse with use. Such a surface is hard to disinfect and is susceptible to filling with milk fat and other solids so that cleaning, essential to prolonged liner life, is impaired. Cracked, damaged or worn liners are difficult to clean and disinfect. They act as reservoirs for mastitis-causing organisms and do not function properly during milking. Liners should be discarded after the number of uses recommended by the manufacturer. They should also be discarded when they lose shape or become rough or cracked.

Liners have to be of adequate length to ensure there is room for collapse beneath the end of the teat to provide effective pulsation. The liner barrel is generally recommended to be no more than about 2 mm wider than the average teat diameter to minimise excessive swelling and stretching of teats.

Liner slip

Liner slip, when the liner loses contact with the teat skin and permits entry of air into the milking system, causes random acute drops in claw vacuum level, and is associated with new IMI of the affected quarter. Typically liner slip produces a 'squawking' sound as air enters through the top of the liner. Once appropriate MM settings are used and clusters are aligned correctly with the cow, liner slip, in turn, is largely a function of liner design and liners without this fault should be used. Liner slipping early in milking may also result from a low vacuum level or blocked air admission holes. In general, a higher rate of liner slips results in a higher rate of new IMI (Rogers and Spencer, 1988, Baxter et al., 1992). Furthermore, unsuitable liners or those not matched to the teat cup may also cause damage to the teat. In the same way, liners which are allowed to lose their resilience and shape through over-use are a serious risk to the herd health Consequentially, it is common practice to assess whether appropriate liners are being used during routine MM maintenance visits.

Milking management factors and their relative effect on mastitis incidence

The commonest management factors mentioned in the literature are: over-milking and under-milking.

Over-milking

Over-milking is almost universally regarded by advisors as an

important factor in machine-induced mastitis. However, while it is undoubtedly inefficient, the evidence does not support a major direct effect on mastitis, particularly when the MM has been adjusted to meet commonly-used standards. For example, although front quarters are over-milked for longer, more mastitis arises in rear quarters (Grindal, 1988).

Over-milking can definitely have an adverse effect on the teats' condition, which in turn, may lead to an increased risk of new IMI (Osteras and Lund, 1988; Edmondson, 2001). Most cows can be milked out in 4 to 6 minutes, yet many MM operators leave the unit on the cow for 2-6 minutes longer (for example when attempting to handle more than the recommended units per person or where problems related to cow flow through the dairy are prolonging the milking time). It is generally accepted that teats over-milked for five minutes show a higher level of damage to the epithelial lining and more extravasation of red blood cells (McDonald, 1971). Over-milking for a long period removes more keratin and causes teat sinus injury (Edmondson, 2001; Gleeson et al, 2003). Extreme periods of over-milking are known to increase teat damage, either at the orifice or to the mucous membrane lining the teat sinus.

Over-milking combined with other faults such as vacuum fluctuations or inadequate pulsation can exacerbate problems (Grindal, 1988), probably by giving a greater exposure of the teat to the deleterious effect. The widespread use of automatic cluster removers has markedly reduced over-milking but, if adjusted incorrectly, may lead to under-milking a proven cause, not of new infections but of increasing the somatic cell count indicating increased reaction to the severity of the infection and possible transition to a clinical case.

Incomplete milk-out (under-milking)

The aim of a good milking routine is to maximize the amount of milk removed from the udder at each milking. Incomplete milk-out has been recognised as a fault for centuries. In 1615, Markham in his book 'Complete Housewife' states that ' the worst point of housewifery that can be is to leave a cow half milked...' (cited from Bramley et al., 1992). Under-milking has been demonstrated to increase the risk of new IMI and severity of the disease in affected quarters (Shepherd, 2000). The large numbers of bacteria and toxins left in the gland after milking impair local defence mechanisms leading to an exacerbation of mammary inflammation. It is likely that the increased frequency of milking that is becoming feasible with the development of automatic milking systems will offer benefits in the resolution of infections or by preventing the progression of a sub-clinical infection to clinical mastitis. The most common causes of poor milk-out are: poor type or condition of liner; clusters that are too light; clusters that do not hang evenly on the udder because connecting tubes are too long, too short, twisted or poorly aligned (Mein et al., 1995); incorrectly adjusted milk flow sensors in case of 'hard milkers'; very low or very high milking vacuum levels; some non-MM factors such as teat lesions; and stray voltage or other stress factors leading to inhibition of milk ejection. Correcting factors that cause under-milking will usually result in a reduction in the bulk milk somatic cell count and in clinical mastitis incidence in problematic herds.

Discussion and Conclusion

Mastitis is a complex production disease resulting from many factors, and there is no simple model that will consider all possible facets. It may be, however, to rank the various risk factors as to their likelihood of causing a problem and, thereby suggest the effort that should be put in to managing the risk of infection that each of these risk factors poses. It is important always to remember that mastitis is multi-factorial, so it is necessary to assess the cost effectiveness of any recommended change and its relative importance in bringing a situation back into control.

The following factors, associated with the MM, are likely to have a major impact on the new IMI rate:

- High vacuum level. An excessively high vacuum level causes teat damage.
- Pulsation failure. The failure may be caused by a fault in the pulsator itself, due to worn out liners, leaks in pulsation tubing, low tension liners, i.e., in an unsuitable teat cup or short effective length liners.
- Over-milking. If excessive, over-milking exacerbates teat damage especially if coupled with high vacuum. It also removes more keratin from the teat canal.
- Liner slip or the equivalent, faulty cluster removal, i.e., without lowering the vacuum inside the claw. The authors advise shutting off the vacuum to the claw and allowing the vacuum to fall for about 1 second before removal - preferably by rotating the claw through about 60 degrees so the liners drop off the teats simultaneously?
- Under-milking. It is probable that under-milking does not directly affect the incidence of new IMI greatly but makes existing infections worse, particularly when the host defence mechanisms are impaired and the entered organisms are in high numbers and/or virulent.
- Unsuitable liners. Liners may affect any of the preceding or following list if they result in incomplete milking, are too short for the cows' teats, are not in the recommended range of tension, are worn or slip.

The following factors should have less impact on the new IMI rate:

1. Vacuum fluctuations. Little effect expected other than those that result in liner slip.
2. Inadequate vacuum pump capacity, faulty vacuum regulation, undersized or unsuitable plumbing all may make cluster changing more difficult and indirectly cause slip or other faults, such as prolonged milking.
3. Reverse flow of milk. This effect will shift infected milk quickly around the MM. There is however no evidence to encourage a trend towards lowline vs. highline milking systems.

The main conclusion of this review is that, in general, MM's have only a limited influence on mastitis occurrence on the farm provided:

- The milking equipment selected for a particular farm operation is that which is best suited for the management, labour, and

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physical facilities available.

- The MM meets internationally recognised standards for installation and performance. The primary concern is that the milking operation does not put unnecessary stress on the cows.
- The MM is regularly maintained, periodically tested and deficiencies corrected.

When dealing with problematic herds, the MM factors are usually just one of the factors in the mastitis epidemiology. During the intervention is important to address also the other factors, such as management, teat-spraying, detection and treatment of new infections.

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